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Dobutamine Stress Echocardiography: Detection of Coronary Artery Disease in Patients With Dilated Cardiomyopathy

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Objectives. **This study attempted to determine the safety and accuracy of dobutamine stress echocardiography for detection of coronary artery disease in patients with dilated cardiomyopathy.**

Background. **Detection of regional wall motion abnormalities at rest does not reliably distinguish ischemic from nonischemic cardiomyopathy. Previous studies have shown that dobutamine stress echocardiography safely and accurately identifies coronary artery disease in patients without dilated cardiomyopathy.**

Methods. **Seventy patients with dilated cardiomyopathy underwent dobutamine stress echocardiography. Echocardiograms were obtained at baseline and at low (5 to 10 μg/kg body weight per min) and peak doses of dobutamine. Rest and stress left ventricular wall motion scores were derived from analysis of regional wall motion. Fifty-four subjects underwent coronary angiography.**

Results. **Dobutamine infusion was terminated after achievement of the target heart rate or maximal protocol dose in 49 patients (70%), ischemia in 12 (17%), arrhythmia in 4 (6%) and side effects in 5 (7%). No patient had prolonged ischemia or sustained arrhythmia. Of those with angiographic studies, 40 had significant coronary artery disease (≥50% diameter stenosis). Use of the change in global wall motion score index from low to peak dose resulted in a sensitivity of 83% for dobutamine stress echocardiography and a specificity of 71% for detection of coronary artery disease. Sensitivity for detection of triple-, double- and single-vessel disease was 100%, 83% and 69%, respectively.**

Conclusions. **Dobutamine stress echocardiography safely provides diagnostic information in patients with dilated cardiomyopathy. This technique has high sensitivity for multivessel coronary artery disease but only moderate specificity.**

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Various noninvasive tests have been evaluated for their ability to detect coronary artery disease in patients with dilated cardiomyopathy (1–14). The detection of coronary artery disease in this group of patients is important for both therapeutic and prognostic reasons (15–21). Echocardiography performed at rest is widely used to evaluate patients with dilated cardiomyopathy. Unfortunately, the frequency of regional wall motion abnormalities at rest in nonischemic cardiomyopathy and the presence of global dysfunction in patients with advanced ischemic cardiomyopathy prevent accurate identification of coronary artery disease (1,2).

Exercise stress testing is the standard noninvasive technique used for the detection of coronary artery disease in patients without dilated cardiomyopathy. Exercise testing is not routinely used for diagnostic purposes in patients with dilated cardiomyopathy because of their reduced ability to perform dynamic stress. Additionally, the frequency of electrocardiographic (ECG) abnormalities at rest impairs interpretation of the stress ECG.

Dobutamine stress echocardiography is an emerging technique that has been found to accurately identify patients with coronary artery disease (22–24). A major advantage of this stress-testing technique is that its accuracy is not dependent on the patient's ability to exercise. Additionally, high quality images can be obtained that permit improved analysis of regional and global systolic function at various degrees of stress. The normal response of the left ventricle to increasing doses of dobutamine in the absence of coronary artery disease is a progressive increase in contractility. In the presence of significant coronary artery disease, contractility may initially improve at low doses of dobutamine, but regional and global function may decline at higher doses that produce myocardial stress and ischemia (22).

In this study, patients who had dilated cardiomyopathy underwent dobutamine stress echocardiography to determine whether stress-induced changes in wall motion could distinguish patients with coronary artery disease from those without. We selected patients with left ventricular dilation and exten-

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sive wall motion abnormalities because, in these patients, assessment of regional wall motion at rest and after standard exercise testing would be less likely to provide useful diagnostic information.

Methods

Patient selection. Between 1988 and January 1992, >1,500 patients underwent dobutamine stress echocardiography at the Indiana University Medical Center. Seventy patients (58 men, 12 women) were included in the study if they had a left ventricular diastolic diameter >5.2 cm, extensive wall motion abnormalities with global wall motion score of ≥1.5 at rest and reduced left ventricular systolic function manifested as a fractional shortening <0.18 or a fractional area change <0.36 (25). Dobutamine stress echocardiographic studies were approved by the Institutional Review Board of the Indiana University Medical Center, and all subjects gave written informed consent.

Dobutamine infusion protocol. Dobutamine was infused at a beginning dose of 5 μg/kg body weight per min and was increased to 10 μg/kg per min after 3 min and then by 10-μg/kg per min increments at 3-min intervals to a peak dose of 30 μg/kg per min. After October 1989, the maximal dose was increased to 50 μg/kg per min. Atropine was administered in 0.25- to 0.35-mg doses in patients with inadequate heart rate responses to dobutamine infusion, as determined by the physician monitoring the test.

Dobutamine infusion was terminated when the maximal protocol dose or a target heart rate of 85% of the patient's age-predicted maximum was achieved. The test was also terminated if angina or significant ST segment depression developed. Induction of wall motion abnormalities was not used routinely as an end point in dobutamine echocardiographic studies performed after October 1989. However, in this group of patients with dilated cardiomyopathy, development of additional wall motion abnormalities in two or more segments remained a reason for test termination. Additional end points included development of significant arrhythmias or side effects.

If demonstrable ischemia occurred, sublingual nitroglycerin or intravenous esmolol (0.25 mg/kg) was administered at the discretion of the investigator.

Stress electrocardiography. Continuous ECG monitoring was performed. A 12-lead system was used in 51 patients, and three bipolar leads were monitored in the remaining 19 patients. The stress ECG was interpreted as normal, ischemic or nondiagnostic. Ischemic changes were defined as the development of ST segment depression ≥1 mm in a lead with a normal baseline or ≥2 mm ST segment depression occurring in the presence of ST segment abnormalities at rest. Stress ECG findings were considered nondiagnostic in the presence of left bundle branch block or digitalis therapy or if ST segment depression <2 mm occurred in the setting of baseline ST segment abnormalities.

Electrocardiographic monitoring was continued for ≥6 min after discontinuation of the infusion.

Stress echocardiography. The four standard echocardiographic views were obtained as previously described (22). Digital images were acquired at rest, with low dose (5 or 10 μg/kg per min), peak dose and 6 min after discontinuation of the infusion. The images were then displayed in a quad screen format that allowed direct comparison of rest and stress images for each view.

The images were interpreted by an investigator who had no knowledge of the clinical history, stress ECG results and angiographic data. In the absence of coronary artery disease, the expected normal response to increasing doses of dobutamine is a progressive increase in wall motion and thickening. Worsening or failure to improve wall motion from the low to peak dose stages of dobutamine infusion was defined as an abnormal result consistent with the presence of significant coronary artery disease.

The left ventricle was divided into 16 segments to evaluate regional wall motion. A global wall motion score index was derived for each stage by summation of all segments scored divided by the number of scored segments for each stage of the examination (25). Regional wall motion score indexes were derived for the territories supplied by the left anterior descending coronary artery (anterior region) and for segments supplied by the left circumflex and right coronary arteries (posterior region) (26). Segmental wall motion was graded on a scale of 1 to 4. A score of 1 represented normal wall motion at rest and hyperkinesia with dobutamine infusion. Segments with hypokinesia at rest received a score of 2. Segments with normal wall motion at rest were considered to have stress-induced hypokinesia if there was a lack of improvement or a reduction in wall motion from rest to low dose or low to peak dose. Akinetic segments were given a score of 3 and dyskinetic segments a score of 4.

Patients demonstrating a progressive improvement in wall motion manifested by a decrease in wall motion score during dobutamine infusion were considered to have nonischemic cardiomyopathy. Patients who had no improvement or worsening of wall motion with dobutamine manifested by no change or an increase in wall motion score from the low to peak dose stages were considered to have ischemic cardiomyopathy.

Coronary angiography. Fifty-four patients (77%) underwent coronary angiography. Visual assessment of the angiograms was initially performed to identify arteries with any degree of stenosis. Manual caliper measurements were then obtained from the cine film image that demonstrated the most severe stenosis by an investigator with no knowledge of the clinical, ECG and echocardiographic data. One angiogram was evaluated by visual estimation because of technical factors that prevented caliper measurement.

Significant coronary artery disease was defined as ≥50% reduction in the absolute lumen diameter of a major epicardial artery or major branch vessel. Patients who had significant narrowing of at least one major coronary artery were considered to have ischemic cardiomyopathy.

Table 1. Medication Profile

	Ischemic DCM (n = 40)	Nonischemic DCM (n = 14)	DCM, No Cath (n = 16)
Calcium channel blockers	13 (32%)	2 (14%)	8 (50%)
Beta-blockers	6 (15%)	0	1 (6%)
Nitrates	29 (73%)*	4 (29%)*	8 (50%)
ACE inhibitors	19 (48%)	10 (71%)	6 (38%)
Digoxin	21 (51%)	6 (43%)	3 (19%)
No therapy	4 (10%)	2 (14%)	2 (13%)
Monotherapy	6 (15%)	3 (21%)	7 (44%)
Two-drug therapy	10 (25%)	7 (50%)	1 (6%)
Three or more drugs	20 (50%)†	2 (14%)†	6 (38%)

*p = 0.005, †p = 0.027. ACE = angiotensin-converting enzyme; Cath = coronary angiography; DCM = dilated cardiomyopathy.

Statistical analysis. Data are given as the mean value ±SD. Descriptive data are given for all three groups. Comparisons were performed only in those patients who had coronary angiography. The Fisher exact test was used to compare categoric variables, and a *t* test was used to analyze continuous variables. Global wall motion scores were compared in the two groups with repeated measures analysis of variance. The grouping factor was the presence or absence of coronary artery disease, and the repeated measure was the dobutamine dose (rest, low and peak). A significant interaction term indicates that the pattern of change with drug is different in the two diagnostic groups. When the interaction term was significant, *t* tests were used to analyze the changes in global wall motion scores between rest and low dose and between low and peak dose to assess more precisely where these changes occurred.

Results

Clinical data. Patients had a mean age of 58 years (range 24 to 78), and 30 patients had a history of congestive heart failure. Twenty-two patients reported chest pain symptoms, and five complained of dyspnea on exertion.

Sixty-two patients (88%) were receiving medical therapy at the time of the study. As shown in Table 1, only the use of nitroglycerin-containing preparations and treatment with multiple medications were more common in the group with coronary artery disease.

Test end points and safety data. The results were evaluated to determine the safety of dobutamine administration in patients with dilated cardiomyopathy. As shown in Table 2, 49 (70%) of 70 studies were terminated after achievement of the target heart rate or the maximal protocol dose. An ischemic end point was achieved in 12 patients (17%). Three patients had severe angina, and in nine patients the test was terminated because of extensive stress-induced wall motion abnormalities. No patient experienced prolonged ischemia. Ischemia was treated with sublingual nitroglycerin in two patients and intravenous esmolol in two others. Resolution of ischemia occurred in the remaining eight patients without additional measures. Ventricular arrhythmias prompted test discontinuation in 4 (6%) of 70 studies. Episodes of ventricular tachycardia longer than three complexes were seen in two patients, and all excess ventricular ectopic activity resolved after discontinuation of the infusion. No patient with ventricular arrhythmia had hypotension. Of the remaining studies, five were discontinued because of dyspnea (n = 3) nausea (n = 1) and back pain (n = 1). All side effects resolved promptly with termination of the infusion.

Table 2. Stress Test End Points

	Ischemic DCM (n = 40)	Nonischemic DCM (n = 14)	DCM, No Cath (n = 16)
THR or max dose	28 (70%)	10 (71%)	11 (69%)
Angina	3 (7%)	0 (0%)	0 (0%)
Wall motion abnormality	5 (13%)	1 (7%)	3 (19%)
Dyspnea	2 (5%)	1 (7%)	0 (0%)
Ventricular arrhythmia	2 (5%)	1 (7%)	1 (6%)
Side effects	0 (0%)	1 (7%)	1 (6%)

max = maximal; THR = target heart rate; other abbreviations as in Table 1.

Patients with coronary angiography. Fifty-four of the 70 patients underwent coronary angiography. Fourteen patients had normal findings or insignificant coronary artery disease. Forty patients had significant coronary artery disease. Single-vessel disease was present in 13 (32%) of 40 patients, two-vessel disease in 18 (45%) and three-vessel disease in 9 (23%). Two of the patients with triple-vessel disease also had significant left main coronary artery disease. As shown in Table 3, the pretest prevalence of angina, history of myocardial infarction and Q waves on the ECG were significantly higher in the group with ischemic cardiomyopathy. Left bundle branch block was more frequent in those with nonischemic cardiomyopathy, and the frequency of heart failure was similar in both groups.

Hemodynamic and ECG data. Table 4 demonstrates the hemodynamic and ECG responses to dobutamine stress testing. Rest and peak heart rates in the group with ischemic cardiomyopathy were significantly lower than those in the group with nonischemic cardiomyopathy. This observation for peak heart rate remained despite correction for the difference in mean age.

The ECG response was nondiagnostic in the majority of patients in both the ischemic and nonischemic groups because of a high frequency of baseline ECG abnormalities. No test was terminated because of ECG changes. Three (7%) of the 40 patients with ischemic cardiomyopathy and 1 (7%) of the 14

Table 3. Clinical and Electrocardiographic Characteristics

	Ischemic DCM (n = 40)	Nonischemic DCM (n = 14)	p Value
Mean (±SD) age (yr)	62 ± 11	48 ± 12	< 0.001
Angina	20 (50%)	2 (14%)	0.027
Prior infarction	27 (68%)	1 (7%)	< 0.001
Heart failure	16 (40%)	9 (64%)	NS
Exertional dyspnea	3 (7%)	1 (7%)	NS
Q waves	20 (50%)	2 (14%)	0.02
Left bundle branch block	4 (10%)	7 (50%)	0.004

DCM = dilated cardiomyopathy.

Table 4. Stress Test Results

	Ischemic DCM (n = 40)	Nonischemic DCM (n = 14)	p Value
Rest HR (beats/min)	78 ± 14	92 ± 20	0.005
Peak HR (beats/min)	122 ± 19	141 ± 14	0.041
Peak systolic BP (mm Hg)	131 ± 31	134 ± 32	NS
Rate-pressure product	16,175 ± 5,083	18,825 ± 5,082	NS
Chest pain	8/40 (20%)	2/14 (14%)	NS
ECG			
Normal	4/40 (10%)	0/14 (0%)	NS
Nondiagnostic	33/40 (83%)	13/14 (93%)	NS
Ischemic	3/40 (7%)	1/14 (7%)	NS

Data presented are mean value ± SD or number (%) of patients. BP = blood pressure; DCM = dilated cardiomyopathy; ECG = electrocardiogram; HR = heart rate.

patients with nonischemic cardiomyopathy demonstrated significant ST segment depression at peak stress.

Analysis of baseline echocardiograms. Mean left ventricular diastolic diameter was larger in patients with nonischemic (6.67 ± 1.20) than ischemic (6.06 ± 0.64) cardiomyopathy (p = 0.02). However, there were no significant differences in the values for fractional shortening (0.14 ± 0.06 vs. 0.13 ± 0.05) and fractional area change (0.24 ± 0.10 vs. 0.21 ± 0.12) in patients with and without coronary artery disease. All of the patients with nonischemic cardiomyopathy had regional wall motion abnormalities. Three of the 40 subjects with ischemic cardiomyopathy had global hypokinesia.

Diagnostic utility of global wall motion scores. The individual and mean global wall motion scores for the two groups of patients are shown in Figure 1. Mean rest global wall motion score was significantly worse in the group with nonischemic cardiomyopathy (2.34 ± 0.42 vs. 2.08 ± 0.35, p = 0.028). There were no significant differences in mean wall motion scores at low and peak dose between the two groups. In patients with ischemic cardiomyopathy the rest global wall motion score demonstrated a trend toward improvement at low dose (1.97 ± 0.42) and then returned to baseline at peak dose (2.05 ± 0.43). In contrast, the patients without coronary artery disease demonstrated a progressive improvement in wall motion score from rest to low (2.14 ± 0.38) to peak doses (1.91 ± 0.43) of dobutamine. The interaction term from the repeated measures analysis of variance was highly significant (p < 0.001). Further analysis indicated that both groups had similar decreases in global wall motion score from rest to low dose (p = 0.418). However, the patients with coronary artery disease showed an increase in score from low to peak dose, whereas the patients without coronary artery disease had decreased scores. This difference in response was significant (p < 0.001).

Evaluation of changes in global wall motion score from low to peak dose correctly identified 33 of 40 patients with and 10 of 14 patients without significant coronary artery disease. Overall sensitivity of dobutamine stress echocardiography for detecting coronary artery disease was 83%; specificity was 71%. The positive predictive value for dobutamine stress echocardiography was 89%; the negative predictive value was 59%.

Of the patients with left main or three-vessel coronary artery disease, nine (100%) of nine were correctly detected. Fifteen (83%) of 18 patients with two-vessel coronary artery disease and 9 (69%) of 13 patients with single-vessel disease were also correctly detected. If lesions >75% were considered, then all of the patients with two-vessel coronary artery disease were correctly identified as having significant disease. All six of the patients with ischemic cardiomyopathy who were receiving beta-adrenergic blocking agents were correctly identified even though none achieved his or her target heart rate.

Four patients in the group with single-vessel disease had false negative findings for ischemic cardiomyopathy because of improvement in global wall motion score from low to peak dose. One patient had total occlusion of the right coronary

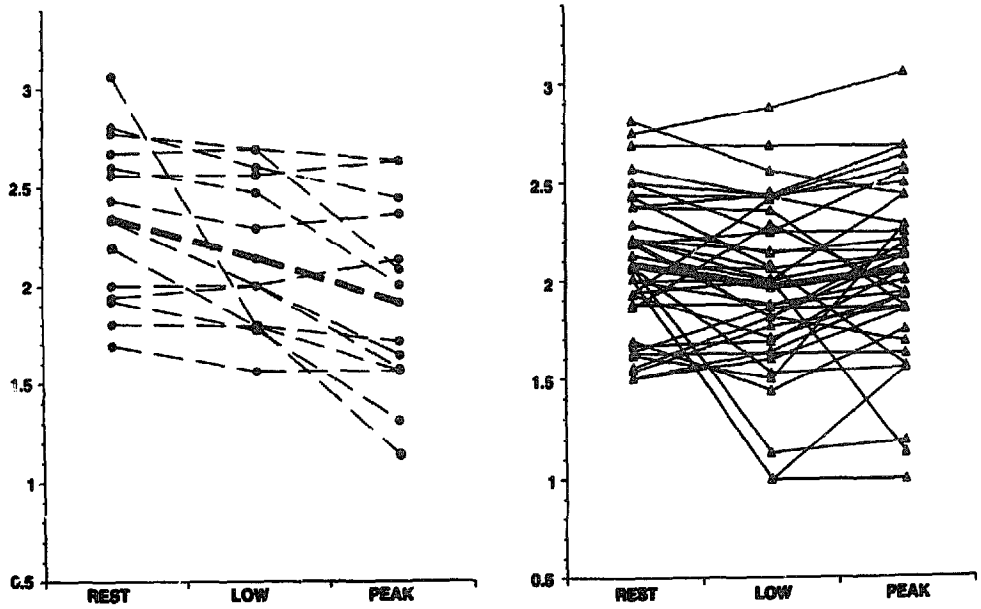

Figure 1. Global wall motion scores for the 14 patients with nonischemic cardiomyopathy (**left**) and the 40 subjects with ischemic cardiomyopathy (**right**). **Bold lines** indicate mean values for each group.

artery but demonstrated extensive left to right collateral vessels. A second patient with a diseased left circumflex artery had some worsening of wall motion in the posterior region but demonstrated improvement in global wall motion score because of greater imrprovement in anterior wall motion. Two other patients had improvement in global wall motion score despite left anterior descending coronary artery stenoses of 50% to 75%.

Three patients with two-vessel coronary artery disease also demonstrated unexpected improvement in global wall motion score. All three had significant disease of the left circumflex and right coronary arteries without involvement of the left anterior descending coronary artery. Two had severe baseline wall motion abnormalities involving the inferoposterior segments. In these patients, wall motion improved in anterior segments, resulting in an overall improvement in global wall motion score. The third patient had a poor response to dobutamine (peak heart rate 96 beats/min).

Four patients with nonischemic cardiomyopathy had unexpected worsening of global wall motion score from low to peak dose. Two of these patients had both severe left ventricular dilation (diastolic dimensions 6.8 and 9.8 cm) and severely reduced systolic function (fractional shortening of 0.06 and 0.11 and fractional area change 0.07 and 0.06). Additionally, these two patients and one other with a false positive study had left bundle branch block. A fourth patient with nonischemic cardiomyopathy and worsening of wall motion from low to peak dose had left ventricular hypertrophy and achieved a high level of stress (peak heart rate 144 beats/min).

Diagnostic utility of regional wall motion scores. The sensitivity for detection of ischemic cardiomyopathy improved to 93% (37 of 40) with analysis of regional wall motion scores. If patients with ischemic cardiomyopathy were identified on the basis of a lack of improvement or worsening of wall motion score in at least one region (anterior or posterior), then four of seven subjects with false negative findings (on the basis of global wall motion scores) would have been correctly diagnosed.

However, assessment of changes in regional wall motion scores would have decreased the specificity for detection of ischemic cardiomyopathy from 71% (10 of 14) to 50% (7 of 14). Global wall motion scores improved from low to peak dose in 10 patients with nonischemic cardiomyopathy, but only 7 of these patients had improvement in both anterior and posterior regions. In the remaining three subjects, anterior wall motion scores demonstrated no improvement.

In the 37 patients with ischemic cardiomyopathy who were correctly identified on the basis of regional wall motion scores, the low to peak dose changes in these scores correctly identified the location of significantly diseased vessels in 55 (74%) of 74 regions.

Discussion

Multiple noninvasive approaches have been used to identify coronary artery disease in patients with dilated cardiomyopathy (1–14). Echocardiographic and nuclear angiographic techniques use analysis of regional wall motion for detection of coronary artery disease. Regional wall motion abnormalities in patients with normal left ventricular dimensions can accurately identify patients with coronary artery disease. Medina et al. (1) demonstrated a 95% sensitivity and 100% specificity for the echocardiographic identification of coronary artery disease in 43 patients with normal left ventricular size and reduced left ventricular systolic function. The presence of regional wall motion abnormalities in patients with dilated cardiomyopathy, however, is not as sensitive or specific. Medina et al. (1) found that the presence of regional wall motion abnormalities in 60 patients with left ventricular dilation had a sensitivity and specificity of only 83% and 57%, respectively, for the presence of coronary artery disease. Others (4,14) have reported that up to two-thirds of patients with nonischemic cardiomyopathy have regional wall motion abnormalities at rest. In our study, all of the patients with nonischemic cardiomyopathy had regional wall motion abnormalities.

In the absence of coronary artery disease, increasing doses of dobutamine produce hyperkinetic wall motion. In the presence of coronary artery disease, the increased work load resulting from high dose dobutamine infusion produces ischemia with worsening of wall motion. This difference in wall motion response enables accurate detection of coronary artery disease in patients without dilated cardiomyopathy (22–24). In general, the low to peak dose changes in global wall motion score were useful for distinguishing patients with ischemic and nonischemic cardiomyopathy. Those patients with more extensive or severe coronary artery disease (left main coronary artery, three-vessel or two-vessel disease with >75% stenosis) were correctly detected with a sensitivity of 100%. However, the sensitivity declined in patients with less extensive disease.

The unexpected improvement in global wall motion score observed in those patients with dilated cardiomyopathy and less extensive coronary artery disease may have several explanations. The left ventricular dysfunction in these patients may have both ischemic and nonischemic causes. In this investigation, there was a higher proportion of single-vessel disease in the ischemic cardiomyopathy group than has been reported in other studies (1,4,9–11). Regions with reduced function but no significant coronary artery disease may demonstrate improvement in wall motion with dobutamine, whereas regions supplied by stenotic coronary arteries may worsen slightly or not at all if these areas have extensive or complete infarction. This situation occurred in three of seven false negative findings and resulted in an improvement in global wall motion score from low to peak dose despite the presence of significant coronary artery disease.

This technique also relied on the demonstration of ischemia for detection of coronary artery disease. The presence of extensive collateral vessels or the lack of functional flow restriction despite angiographic narrowing could prevent ischemia and result in unexpected improvement in wall motion. Additionally, failure to attain sufficient myocardial stress to produce ischemia may result in false negative findings. In dilated cardiomyopathy, dobutamine administration may reduce preload and afterload, limiting the induction of ischemia.

The specificity of dobutamine stress echocardiography in this study was lower than that reported in patients with normal

rest wall motion (22–24). There are several possible explanations for the false positive results obtained in the group with nonischemic cardiomyopathy. Some of these patients may have myopathic disease of such an advanced stage that the myocardium could no longer respond to dobutamine stimulation. Both left ventricular diastolic diameter and rest global wall motion score index were significantly worse in the group without coronary artery disease. Additionally, reduced coronary flow reserve has been observed in some patients with dilated cardiomyopathy and may result in stress-induced myocardial ischemia even in the absence of epicardial coronary artery disease (27,28).

Three patients with false positive findings had left bundle branch block. Left bundle branch block results in asynchronous septal contraction, which may alter septal perfusion and produce ischemia in the absence of epicardial coronary artery disease (29,30).

Study limitations. The number of patients with nonischemic cardiomyopathy was small, so that any conclusions with regard to the specificity of dobutamine stress echocardiography must be made with caution. Differentiation of ischemic and nonischemic cardiomyopathy solely on the basis of coronary angiography is imprecise because more than one process may be present simultaneously, and angiography does not provide a physiologic assessment of the severity of coronary artery disease. Finally, analysis of wall motion was limited to qualitative techniques.

Conclusions. Dobutamine stress echocardiography is a widely available technique that can safely provide diagnostic information in patients with dilated cardiomyopathy. The wall motion changes induced with progressive doses of dobutamine identified patients with extensive coronary artery disease with a high sensitivity. However, only a modest specificity was achieved by evaluation of changes in global wall motion score.

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